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Neurogenic pulmonary oedema in a patient with leptomeningeal carcinomatosis

We present the case of a patient with leptomeningeal carcinomatosis from a primary breast adenocarcinoma. On admission to hospital, she developed neurogenic pulmonary oedema due to increased intracranial pressure (ICP), as confirmed by lumbar puncture. Tapping of the CSF resulted in abrupt relief of the clinical sequelae.

A woman in her late 40s, with a medical history of breast adenocarcinoma, for which she underwent a modified radical mastectomy with axillary lymph node resection, was admitted to our hospital for intrathecal chemotherapy. Just 1 month earlier, she underwent craniotomy for a solitary metastatic intracerebral lesion in the left frontal lobe. After this uneventful procedure, she continuously complained of drowsiness, nausea and vomiting. A CT scan showed enhancement of cerebral and cerebellar sulci, typical of leptomeningeal metastasis.

This diagnosis prompted admittance to our hospital, a tertiary care oncology centre, for

further treatment. On admittance, the patient had increasing dyspnoea. Pulmonary and cardiac examination showed basilar crepitations on both lungs, tachypnoea (>24/min), tachycardia (145 beats/min) and a blood pressure of 80/40 mm Hg. No cardiac murmurs were heard and the electrocardiograph showed no signs of myocardial infarction. The peripheral oxygen saturation at that time was 80% with no oxygen support. Blood gas analysis showed hypoxia: pH 7.46, partial pressure of carbon dioxide (pCO₂) 4.7, partial pressure of oxygen (pO₂) 5.4, HCO₃⁻ 24.7, (BE) 1.3 and arterial oxygen saturation (SaO₂) 0.79. A chest radiograph showed a normal heart with evidence of bilateral alveolar filling (fig 1B; fig 1A shows the chest radiograph taken 1 day earlier, which was without abnormalities).

The patient was admitted to the intensive care unit and respiration was supported by mechanical ventilation through a facemask and administration of oxygen. Nevertheless, her neurological condition deteriorated to a Glasgow Coma Scale (GCS) score of 6. On reconsidering the CT scan and the diagnosis of leptomeningeal carcinomatosis, we hypothesised that increased ICP, due to a CSF resorption disturbance, caused neurogenic pulmonary oedema. Therefore, we carried out a lumbar puncture, showing an increased CSF pressure of 60 cm H₂O. We drained 30 ml of the CSF, during which the patient regained consciousness acutely (GCS 13). Cytology showed the presence of malignant cells. The tachypnoea and tachycardia resolved, although the basilar crepitations could still be recognised on pulmonary auscultation. Blood gas analysis showed recovery: pH 7.42, pCO₂ 5.2, pO₂ 8.0, HCO₃⁻ 24.8, BE 1.0 and SaO₂ 0.93.

The patient was readmitted to the neurosurgical department for a CSF shunt procedure. She was, however, not fit for surgery, and attempts to alleviate the increased ICP with an external lumbar drain failed. Treatment was discontinued when her condition deteriorated again 5 days later, after which she died.

As far as we know, leptomeningeal metastasis has not been described as the cause of neurogenic pulmonary oedema. Although the association of an increased ICP and neurogenic pulmonary oedema has been described previously, the pathophysiological mechanisms remain obscured.^{1–3} The medulla oblongata is believed to be the critical anatomical structure responsible for the pathogenesis of

neurogenic pulmonary oedema, probably acting through the sympathetic component of the autonomic nervous system.^{1,2}

The rapid improvement after the normalisation of the increased CSF pressure is more consistent with neurogenic lung oedema than with other causes of respiratory distress. As reviewed by Fontes *et al*,⁴ the clinical features as described in our patient—that is, tachypnoea, dyspnoea, tachycardia and hypotension—often occur in acute neurogenic pulmonary oedema. As Fontes *et al* conclude, we believe that doctors should always be aware of this clinical condition after neurological events, especially when no other causes of pulmonary oedema can be established. Supportive measures are the treatment of choice. In our patient, relieving the increased ICP corrected the respiratory failure.

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Onset of cluster headache triggered by emotional effect: a case report

Cluster headache is a strictly unilateral headache that occurs in association with autonomic symptoms. Stress is a recognised precipitant of migraines, but not of cluster attacks. We describe the case of a patient having migraine for years, in whom extreme emotional stress triggered cluster headache attacks.

Background

Neuroimaging has contributed considerably to the knowledge about the neurobiology of cluster headache. Activation in the region of the posterior hypothalamus is observed,¹ coinciding with subtle structural abnormalities in the same region.²

Genetic studies indicate an autosomal dominant inheritance with low penetrance and an increase in risk of 4–18 times for

Figure 1 Chest radiograph: (A) taken 1 day earlier; (B) with evidence of bilateral alveolar filling.